GENETIC VARIABILITY FOR DENSITY SENSITIVITY OF THREE COMPONENTS OF FITNESS IN *DROSOPHILA MELANOGASTER*†

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ABSTRACT

This study examines natural genetic variation in density sensitivity of three components of fitness in *Drosophila melanogaster* using the method of chromosome extraction. Different lines are differentially sensitive to density. The distribution of measures of density sensitivity of chromosomal homozygotes is different from that of random chromosomal heterozygotes for both location and dispersion. Density sensitivity of the components is about as variable as any of the fitness components themselves at fixed densities. The consequences of the exact nature of this density dependence are discussed with respect to the stage of the life cycle at which density dependence occurs, and the mathematical form that it takes. There is no evidence of trade-offs among the components or their density sensitivity.

THE purpose of this study is to assay the extent of genetic variability in *Drosophila melanogaster* for the sensitivity of several components of fitness to larval density. It is a survey of variation freshly taken from one natural population, using DOBZHANSKY's classical method for assaying genetic variation, chromosome extraction.

Density sensitivity of fitness components has been of interest to experimental population biologists for some time. Many studies have directly examined the influence of density on fitness, fitness components or the outcome of competition between genetically different strains of, as examples, houseflies (SOKAL and SULLIVAN 1963; BHALLA and SOKAL 1964; TAYLOR and SOKAL 1973), Tribolium (SOKAL and HUBER 1963; SOKAL and KARTEN 1964), and Drosophila (HARNLY 1929; BIRCH 1955; LEWONTIN 1955; MOREE and KING 1961; LEWONTIN and MATSUO 1963; DRUGER and NICKERSON 1972; DEBENEDICTIS 1977).

Other studies have looked indirectly at the variability of density sensitivity in various organisms. For example, SOLBRIG (1971), in a study of r- and K-selection, looked at the effect of environmental perturbations on the genetic composition of several populations of dandelions. Genetic differences in density sensitivity of development rate were implied by the selection responses he observed. A similar sort of variation is implied by the studies of AYALA (1968) on intraspecific competition. Some evaluation of genetic variability for density sensitivity is often included in studies aimed principally at some other hypoth-

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esis. As an example, studies of frequency dependent selection often incorporate different experimental densities as well as frequencies (for example, KOJIMA and HUANG 1972; see AYALA and CAMPELL 1974, for a review).

These studies established that density dependence occurs, but did not measure the extent of genetic variation in nature. Usually they focused on only a few strains, and these were often laboratory stocks carrying morphological markers. As examples, SOKAL and colleagues investigated density effects in strains of eye color and body color mutants in houseflies, and body color in Tribolium; LEWONTIN and MATSUO (1963) worked with wing shape, body color, and eye color mutants in *Drosophila buskii*; DEBENEDICTIS (1977) worked on several fourth chromosome morphological markers in *D. melanogaster*. Generally at most one or two wild-type strains were included in these studies.

BIRCH (1955) and DRUGER and NICKERSON (1972) looked at density effects on marker free strains with different inversions in *D. pseudoobscura*. However, their studies were limited to very few lines, and therefore gave no information about the natural distribution of density effects.

These earlier studies establish what is possible, but do not tell us what is probable. The only study that gives evidence about the distribution of density dependence is that of LEWONTIN (1955), although its main emphasis was the effect of genotype interaction of viability. For several reasons, LEWONTIN's work does not give a completely satisfying description of natural variation for density dependence. First, he used chromosomes extracted from flies from population cages, which may not be representative of those found in nature. Second, his study was over a limited range of densities. Third, he looked only at a single component of fitness, egg to adult viability, when studying the sensitivity to density in pure cultures.

In contrast to experimental work, density sensitivity of fitness has been of more recent interest to theoretical population biologists. Several authors have explored models of density dependent selection (ANDERSON 1971; ROUGHGARDEN 1971, 1976) and density independent selection in a density regulated population (CHARLESWORTH 1971; PROUT 1975, 1980). These are theories of evolution, and as all evolutionary theories, make the assumption that the relevant variation exists. A necessary, though perhaps not sufficient, condition for density dependent selection is the existence of genetic variation in the density sensitivity of fitness components. Experimental exploration of these evolutionary models must first establish the existence of this variation.

Surveys of the genetic variation relevant to specific evolutionary hypotheses have used chromosome extraction to magnify that variation. This technique was first applied extensively to natural populations by DOBZHANSKY. It has subsequently been used in surveys of several species of Drosophila and the mosquito, *Culex tritaeniorhynchus* (SAKAI and BAKER 1972). The results of these studies have given us information about the extent of hidden variation in a wide variety of fitness components (summaries in DOBZHANSKY 1970, chapters 3 and 4; and LEWONTIN 1974, chapter 2).

This study, using the method of chromosome extraction, is an investigation into the nature and extent of genetic variability of density sensitivity of viability, development rate, and female fertility.

MATERIALS AND METHODS

Drosophila melanogaster were collected near Berkeley, California, in the fall of 1976. Males from this collection were immediately mated to females from a balanced second chromosome stock (Cy(SM5)/Bl L^2), and second chromosomes extracted by the usual scheme (LEWONTIN 1974, Figure 2). Heterozygous flies from the penultimate step in the extraction were backcrossed to the balancer three times to make the genetic background more uniform from line to line. When needed for the experimental scheme, wild-type virgins were collected and mated to wild-type males of the same or another line. Twenty homozygous lines and ten random heterozygotes were used in the experiment. All stocks were maintained at 25° ± 1° and constant humidity.

Females from these lines were allowed to oviposit for 12 hours on plastic spoons filled with medium colored carbon (day one). After the females were removed, the spoons were placed in the incubator. After 24 hours, different numbers of first instar larvae were collected from these spoons and placed into six-dram vials filled with 8 cc of standard agar-cornmeal-corn syrup-molasses medium (day two). The densities used and minimum numbers of replicates are given in Table 1.

From day seven on, adult flies were collected daily from each of these experimental vials, counted, and placed in fresh vials kept in the incubator. After two days in the fresh vials, females were weighed *en masse* and discarded. The raw data for a particular experimental vial, therefore, consist of the daily emergence numbers and total weight of females for each day.

RESULTS

The experiment reported here takes the fitness components approach (PROUT 1971), in this case examining the dependence of larval survival, development rate, and female fertility on larval density.

These three components of fitness are calculated for each experimental vial as follows:

$$\text{viability} = \frac{\text{total numbers emerged}}{\text{initial larval numbers}}$$

$$\text{development rate} = \frac{\Sigma n(i)}{\Sigma n(i) \cdot i}$$

in which $n(i)$ represents the number of flies emerging on day i. Female body weight is used as a measure of female fertility. In a very careful experiment, BARKER and PODGER (1970) showed that fertility is strongly correlated with body weight; from their Figure 2 I calculate $r = 0.98$ for *D. melanogaster*. The actual body weight data present a bit of a problem, since the raw data are the

TABLE 1

Densities and minimum number of replicates. For some lines, vials were made with 240 larvae

Density	10	20	40	80	160	320
Replicates	10	10	10	5	5	3

total weights of varying numbers of females. The individual data points, therefore, are difficult to interpret properly, as each is based on a different sample size. It is also difficult to get an estimate of the variance of weight: at low densities because the sample sizes vary greatly (proportionally) and at high densities because the individual weights are markedly non-normal. Therefore, in the analyses that follow, I have used the grand mean of body weight for a particular density rather than the individuals observations. The grand means for each fitness component for each line are shown in Table 2.

Are fitness components of different genotypes differentially sensitive to density? This question is answered by looking at the interaction term in a two-way analysis of variance. The results of such analyses of variance for larval survival and development rate are presented in Table 3. (An analysis of variance for weight is not possible for the reasons discussed above.) In each case, the F for interaction is highly significant, indicating genetic variation in density sensitivity of the indicated fitness component.

A more thorough examination of the nature and extent of this variation will require a simple characterization of the sensitivity of the various fitness components to density. The most meaningful measure is the density sensitivity parameter of a biologically reasonable model of density sensitivity. Consider models of the form:

$$N' = N \cdot D(N) \tag{1}$$

in which N measures population numbers. $D(N)$ is called the density regulating function (PROUT 1975). Three different two-parameter models are commonly used:

$$D(N) = (a - bN) \tag{2}$$

$$D(N) = a/(1 + bN) \tag{3}$$

$$D(N) = a \cdot \exp(-bN) \quad . \tag{4}$$

In each case the parameter a is the maximum growth rate, and b measures density sensitivity. Function (2) is derived from the differential logistic equation. Function (3) has been called the logistic difference equation (MAY 1975). PIELOU (1969) demonstrated its mathematical kinship with the logistic, and POULSEN (1975) (cited in CHRISTIANSEN and FENCHEL 1977) derived it from biological first principles. Function (4), proposed by MACFADYEN (1963), was investigated theoretically by MAY (1975), who justified its use by citing its frequent fit to data in the entomological literature.

These three models of density regulation are linearized, respectively, by making no transformation, an inverse transformation, and a log transformation. Each can, therefore, easily be fit to the data for any fitness component by making the appropriate transformation and fitting the transformed data to a linear model. The slope, b, of the fitted model will be used to characterize the density sensitivity.

TABLE 2

Means of indicated components of fitness for indicated density

Line	Density 10	20	40	80	160	320
a. Larval survival						
1	0.8700	0.8000	0.7625	0.7676	0.7239	0.4613
2	0.7200	0.7700	0.7725	0.7274	0.6060	0.4945
6	0.8600	0.8650	0.8091	0.8675	0.7400	0.6596
7	0.8533	0.8300	0.8075	0.7700	0.6893	0.5938
8	0.9300	0.9000	0.9275	0.7751	0.6662	0.4467
9	0.8900	0.8750	0.7150	0.6775	0.5312	0.4588
10	0.8500	0.6350	0.6975	0.7150	0.6050	0.4937
11	0.6900	0.6700	0.6025	0.5375	0.5450	0.3969
14	0.9462	0.9350	0.8675	0.8313	0.7188	0.4425
15	0.7500	0.7950	0.8075	0.6583	0.5788	0.4094
16	0.8200	0.7850	0.8400	0.8400	0.6863	0.4813
17	0.6400	0.6300	0.6100	0.4650	0.3988	0.3302
21	0.8800	0.9000	0.8375	0.8800	0.6525	0.4688
22	0.7600	0.7200	0.7083	0.6899	0.6813	0.4365
23	0.9300	0.8500	0.8300	0.8050	0.7578	0.3573
24	0.9500	0.9400	0.9125	0.8575	0.7264	0.6044
25	0.8300	0.8350	0.7750	0.7300	0.7099	0.4737
27	0.8100	0.8000	0.7975	0.6500	0.6266	0.5538
33	0.8700	0.8700	0.8425	0.7775	0.7188	0.6563
1/2	0.9933	0.9800	0.0925	0.9150	0.8700	0.6896
6/7	0.9500	0.8650	0.8775	0.8813	0.8450	0.6406
8/9	0.9933	0.9500	0.9400	0.9188	0.7950	0.7613
10/11	0.9400	0.9500	0.9150	0.8495	0.7738	0.6994
14/15	0.8900	0.9400	0.9725	0.9275	0.8263	0.7613
16/17	0.9800	0.9750	0.9750	0.9375	0.8775	0.7258
21/22	0.9933	0.9750	0.9650	0.9100	0.9325	0.6698
23/24	0.9800	0.9650	0.9375	0.8750	0.7950	0.6292
25/27	0.9867	0.9700	0.8925	0.8925	0.7863	0.6484
17/33	0.9500	0.9400	0.9150	0.8156	0.6888	0.5863
b. Development rate						
1	0.0962	0.0959	0.0948	0.0887	0.0843	0.0683
2	0.0999	0.0980	0.0961	0.0898	0.0828	0.0689
6	0.1015	0.0997	0.0985	0.0955	0.0878	0.0728
7	0.0955	0.0914	0.0875	0.0779	0.0747	0.0590
8	0.0967	0.0957	0.0921	0.0944	0.0759	0.0621
9	0.0978	0.0964	0.0944	0.0952	0.0751	0.0692
10	0.0983	0.0995	0.0973	0.0889	0.0942	0.0712
11	0.1008	0.0965	0.0938	0.0878	0.0757	0.0611
14	0.0986	0.0988	0.0986	0.0884	0.0813	0.0685
15	0.0994	0.0980	0.0946	0.0892	0.0808	0.0656
16	0.0955	0.0971	0.0992	0.0914	0.0831	0.0702
17	0.0979	0.0964	0.0917	0.0841	0.0744	0.0599
21	0.0994	0.0996	0.0974	0.0922	0.0843	0.0669
22	0.0948	0.0927	0.0902	0.0840	0.0810	0.0657

TABLE 2—Continued

Line	Density 10	20	40	80	160	320
23	0.0995	0.1002	0.0966	0.0923	0.0857	0.0614
24	0.0947	0.0933	0.0885	0.0846	0.0730	0.0611
25	0.0938	0.0932	0.0906	0.0843	0.0821	0.0642
27	0.0996	0.0980	0.0942	0.0898	0.0789	0.0652
33	0.1019	0.1003	0.0970	0.0950	0.0871	0.0727
1/2	0.1038	0.1028	0.1017	0.0951	0.0869	0.0744
6/7	0.1027	0.1019	0.0984	0.0979	0.0863	0.0746
10/11	0.1050	0.1026	0.0957	0.0938	0.0855	0.0728
14/15	0.1029	0.1020	0.0998	0.0985	0.0880	0.0745
16/17	0.1013	0.0990	0.0961	0.0914	0.0814	0.0708
21/22	0.1008	0.0994	0.0960	0.0914	0.0818	0.0650
23/24	0.1045	0.1026	0.0989	0.0938	0.0847	0.0694
25/27	0.1033	0.1022	0.0990	0.0931	0.0833	0.0693
17/33	0.1005	0.0996	0.0959	0.0911	0.0805	0.0682
c. Female body weight						
1	1.215	1.224	1.174	1.136	0.841	0.721
2	1.224	1.184	1.170	1.059	0.933	0.742
6	1.227	1.215	1.154	1.054	0.964	0.781
7	1.140	1.094	1.040	0.960	0.948	0.660
8	1.183	1.147	1.083	1.035	0.937	0.862
9	1.359	1.277	1.320	1.236	0.924	0.814
10	1.203	1.189	1.096	0.997	0.895	0.690
11	1.372	1.358	1.248	1.110	0.924	0.753
14	1.456	1.173	1.087	0.982	0.935	0.664
15	1.318	1.228	1.162	1.114	0.825	0.621
16	1.121	1.184	0.995	0.866	0.798	0.692
17	1.096	1.040	0.965	0.879	0.752	0.552
21	1.200	1.136	1.111	1.057	0.888	0.711
22	1.172	1.057	1.005	0.930	0.745	0.662
23	1.274	1.168	1.062	0.974	0.892	0.762
24	1.131	1.076	1.006	0.948	0.769	0.562
25	1.230	1.079	1.020	1.003	0.850	0.671
27	1.236	1.226	1.169	1.006	0.974	0.781
33	1.215	1.106	1.091	1.063	0.879	0.701
1/2	1.319	1.335	1.309	1.206	1.054	0.857
6/7	1.367	1.378	1.260	1.210	1.055	0.876
8/9	1.316	1.212	1.209	1.128	0.952	0.790
10/11	1.260	1.214	1.245	1.205	1.049	0.838
14/15	1.423	1.317	1.281	1.223	1.076	0.861
16/17	1.418	1.348	1.234	1.164	0.991	0.827
21/22	1.440	1.296	1.248	1.208	1.046	0.808
23/24	1.217	1.210	1.177	1.029	0.984	0.790
25/27	1.406	1.359	1.313	1.276	1.056	0.872
17/33	1.236	1.295	1.194	1.127	1.054	0.872

Line numbers are given at left: single numbers denote chromosomal homozygotes; numbers separated by slash denote chromosomal heterozygotes formed by crossing the two indicated homozygotes.

TABLE 3

Two way analysis of variance of larval survival and development rate for chromosomal homozygotes and heterozygotes

Source	SS	DF	MS	F
a. Larval survival—homozygotes				
Genotype	3.33618	17	0.19625	23.43
Density	8.63178	5	1.72636	206.10
G × D	1.61819	85	0.01904	2.27
Error	6.00573	717	0.00838	
Totals	19.59188	824		
b. Larval survival—heterozygotes				
Genotype	0.31095	9	0.03455	16.96
Density	3.12894	5	0.62579	307.11
G × D	0.36796	45	0.00818	4.01
Error	0.81711	401	0.00204	
Totals	4.62496	460		
c. Development rate—homozygotes				
Genotype	154.7878	17	9.10517	42.70
Density	1689.1311	5	337.82621	1584.30
G × D	76.0727	85	0.89497	4.20
Error	152.8884	717	0.21323	
Totals	2072.8800	824		
d. Development rate—heterozygotes				
Genotype	25.4830	9	2.83145	41.02
Density	706.6010	5	141.32021	2047.51
G × D	12.7525	45	0.28339	4.11
Error	27.6772	401	0.06902	
Totals	772.5138	460		

All values of F are significant at the 0.1% level.

The general scheme for further analysis will be: 1) fit the data to the three models; 2) on the basis of the relative goodness of fit, select one model for use in characterizing the data; 3) examine the estimated density sensitivity parameters (b) for information about the extent of genetic variation.

Each of the three models [equations (2), (3), and (4)] has been fit individually to the means for each density of each component for every homozygous and heterozygous line. In Table 4 are shown the values of R^2, the fraction of the total variance explained by regression, averaged separately for homozygotes and heterozygotes and separately for each component of fitness. Also shown in Table 4 are the sums over lines of the ranks of the values of R^2 within lines.

TABLE 4

Mean R^2 *and rank sums across lines for the indicated transformations and components of fitness*

	Transformation					
	None		Inverse		Log	
	Mean	Rank sum	Mean	Rank sum	Mean	Rank sum
Larval survival						
Homozygotes	0.898	50	0.916	32	0.912	38
Heterozygotes	0.937	23	0.949	19	0.945	18
Development rate						
Homozygotes	0.972	47	0.976	36	0.977	37
Heterozygotes	0.984	28	0.996	14	0.992	18
Weight						
Homozygotes	0.921	58	0.962	22	0.947	40
Heterozygotes	0.957	28	0.985	12	0.975	20

Arbitrarily, because it generally has the smallest rank sum, the functional relationship of each component to density will be characterized by the slope of a linear fit after inverse transformation of the data. To illustrate, in Figure 1 are shown the data for two representative lines, one fairly density sensitive and one fairly density insensitive, and their fitted curves. The untransformed fitted curves for each line and for each component of fitness are shown collectively in Figure 2.

To characterize the nature of the variation I will examine two different attributes: variability of the components themselves, and variability in their density sensitivity. This variation may manifest itself in two different ways: relative to the distribution of random heterozygotes, the distribution of homozygotes may have different variance or different mean or median. The usual

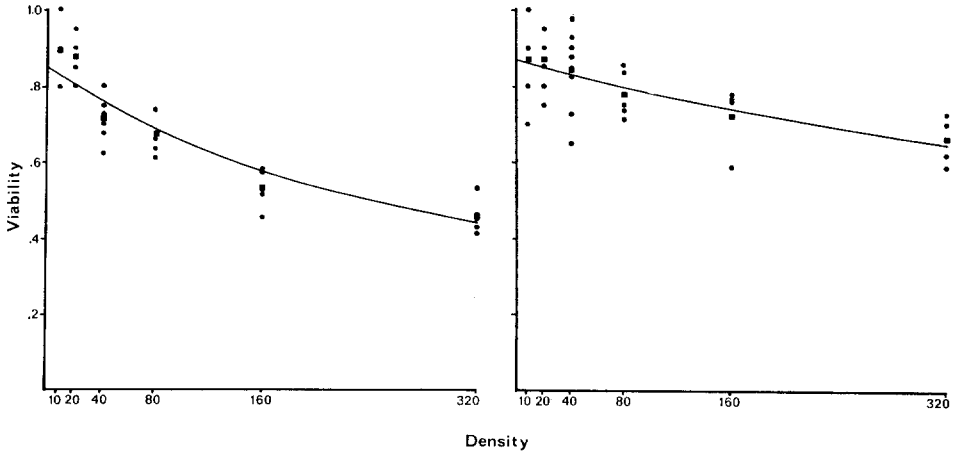

FIGURE 1.—Examples of the fit of the inverse model of density regulation: viability data for lines 9 (left) and 33 (right). Circles are the individual data points; squares are the means for the indicated density.

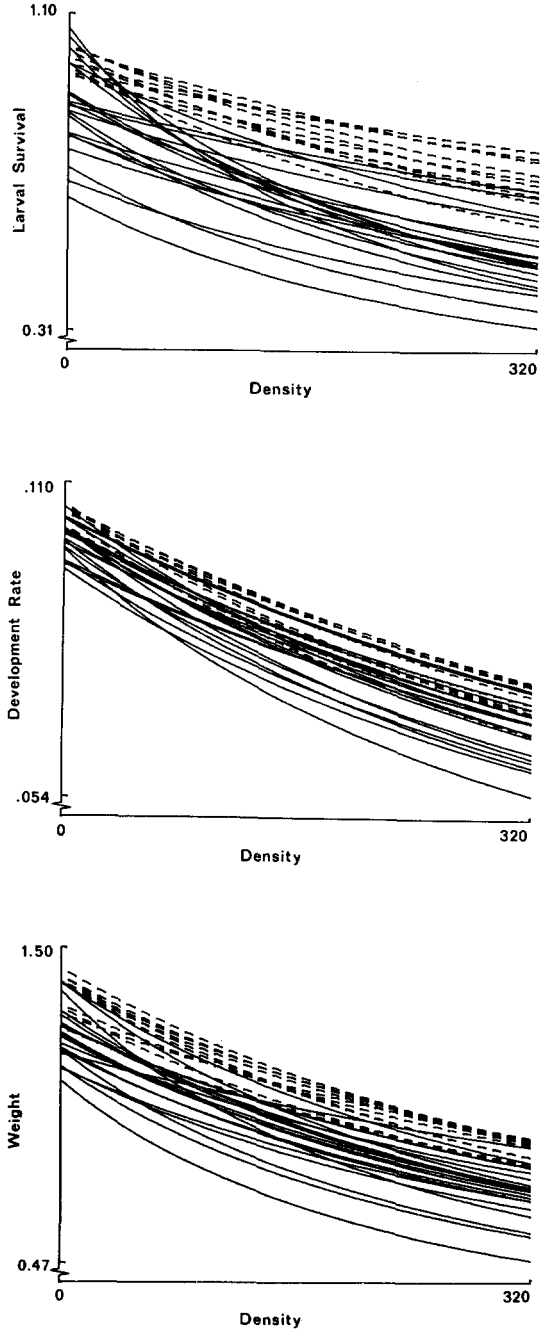

FIGURE 2.—Plots of the fitted hyperbole for the indicated component. Solid lines are homozygotes, dashed lines are heterozygotes.

result of studies of extracted chromosomes is that homozygotes have a different mean and larger variance than heterozygotes (see LEWONTIN 1974, Figure 4).

Tests for variability of the components themselves were made on values of the components estimated from the fitted curves, as this procedure uses all the data. The results are shown in Table 3. The tests were carried out at several densities, as it is clear from Figure 2 that the mean and variance of the components depend on density. In all cases but one, the values of F are significant at least at the 5% level. The extraction scheme has uncovered genetic variation as revealed by significantly larger variance among homozygotes as compared to heterozygotes. In all cases, the value of W, the WILCOXON test statistic for differences in location, is significant at least at the 1% level. Thus, genetic variation has also been revealed by a shift of the mean homozygotes as compared to heterozygotes. These results are in complete agreement with those of previous studies done at single (or more or less uncontrolled) densities (summary in LEWONTIN 1974, pp. 38–66).

The main purpose of this study is to assess the extent of genetic variation in the sensitivity of these components to density. The density sensitivity is measured by the fitted slope parameter, b. Small values indicate density insensitivity; large values indicate density sensitivity. The estimated values of b would customarily be presented in a histogram; in this study, as there are only 20 homozygotes and 10 heterozygotes, a conventional histogram would not be particularly illuminating. The data are shown individually, therefore, in Figure 3: each value of b is shown with its standard error, ranked in order of increasing density sensitivity, homozygotes and heterozygotes separately. The analytic approach to density sensitivity is exactly as above: test whether the distributions of the b's for homozygotes and heterozygotes differ in dispersion (F test) and location (WILCOXON test).

Table 6 shows the means and standard deviations of these distribution for each of the three components. The F values in the table are calculated as the

TABLE 5

Test statistics for tests of difference in dispersion and location of distribution of indicated fitness component at indicated densties

		Densities		
		0	60	320
Larval Survival	F	29.73‡	28.46‡	9.75‡
	W	93†	56‡	62‡
Development Rate	F	2.95*	3.60*	3.83*
	W	79‡	78‡	92†
Weight	F	2.82	5.20†	18.28‡
	W	76‡	63‡	61‡

* $P < 0.05$; † $P < 0.01$; ‡ $P < 0.001$.

F is the ratio of variance of homozygotes to variance of heterozygotes. Significantly large F indicates greater variance among homozygous lines. W is the rank sum for heterozygotes; significantly small W indicates a difference in location of the distributions of homozygotes and heterozygotes.

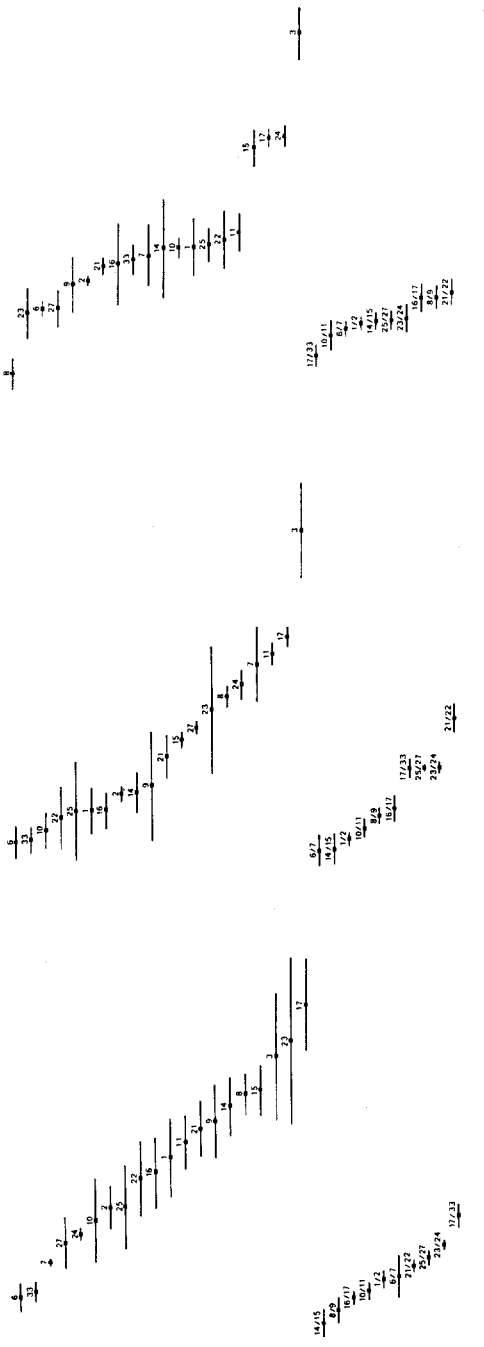

FIGURE 3.—Display of the distribution of the slopes (density sensitivity) of the fits of the data to a linear model after inverse transformation. The midpoint of each line, indicated by a square, is the estimate of the slope; the span is two standard errors. The lines are presented, from top to bottom, in order of increasing density sensitivity (slope), homozygotes followed by heterozygotes, each identified by number (for example, 14/15 is the heterozygote formed by crossing lines 14 and 15). The actual values of the slopes, whose range is given on the abcissa, have been multiplied by: 10^4 for larval survival, 10^2 for development rate, and 10^7 for weight.

ratio of the variance in the slopes among homozygotes to the variance in slopes among heterozygotes. The significantly large values of F (all $p \leq 5\%$) indicate that there is significantly more variance in the sensitivity to density among homozygotes.

The far right-hand column of Table 6 gives the values of the WILCOXON rank-sum. Homozygotes are on the average more density sensitive than heterozygotes with respect to larval survival and body weight, although not with respect to development rate. In general the overall picture for the components' density sensitivity is very similar to the usual results for the components themselves: the extraction scheme has uncovered substantial variability by both the mean and the variance criteria.

DISCUSSION

This study has examined the extent and nature of genetic variability for several components of fitness and for their sensitivity to density. With respect to the components themselves at given densities, the chromosome extraction scheme has revealed variability, both as an increase in the variance among and shift of the mean of homozygous lines compared to heterozygous lines. Previous studies of these same components have had identical results: these lines are not aberrant. The extent of variation in the components themselves establishes an internal benchmark against which the amount of genetic variation in density sensitivity may be evaluated.

The new aspect of this study is the thorough investigation of the sensitivity to density of these several fitness components. LEWONTIN (1955) did a similar experiment looking at viability only, but at densities much lower than those reported here. The lines used in his experiment were taken from laboratory population cages and had previously been scored as subvital or semilethal (viability strictly less than that of the average of random heterozygotes); I have used 20 homozygous lines of chromosomes freshly extracted from a natural population. In addition, LEWONTIN combined heterozygotes as a single control, thus not allowing the quantification of exposed variation done here.

TABLE 6

Means and standard deviations of slopes from line fitted after inverse transformation

Trait	Homozygotes		Heterozygotes			
	Mean	S.D.	Mean	S.D.	F (19,9)	W
Larval Survival ($\times 10^3$)	2.8971	1.0557	1.4249	0.4048	6.80†	80‡
Development Rate ($\times 10^2$)	1.6323	0.3459	1.4047	0.1822	3.60*	118
Weight ($\times 10^7$)	20.1392	5.9739	14.1696	1.5111	15.63‡	93†

* $P < 0.05$; † $P < 0.01$; ‡ $P < 0.001$.

F is calculated as the ratio of the variance of homozygotes to the variance of heterozygotes. The Wilcoxon Rank-Sum is given in the column marked W. The power of ten by which each of the means and standard deviations in the associated row have been multiplied is indicated.

This study has demonstrated significant genetic variation for density sensitivity of all components examined. The analyses of variance (Table 3) show that homozygous lines are differentially sensitive to density, as are heterozygous lines. Substantial genetic variation has been uncovered by the extraction scheme: the mean and variance of the distributions of density sensitivity of homozygotes are different from those of heterozygotes.

Density sensitivity seems to have about as much hidden genetic variation as the components themselves. As a rough measure, we can compare the size of the test statistics obtained from comparisons of homozygotes to heterozygotes. The appropriate values are in Tables 5 and 6. The F ratios of variances and W's for differences in location of density sensitivity are neither obviously smaller nor obviously larger than those for the components at any particular density.

It is important to remember that the data reflect the effect of *larval* density on the three components of fitness. For example, I have shown an effect on female fertility, as reflected by body weight, of the larval density from which these females emerged. This "feed forward" effect differs from that found by CHIANG and HODSON (1950), who demonstrated a slight effect of adult female density on female fertility. A feed forward effect has been found by earlier workers (BAKKER 1961; SANG 1950). It has been explored theoretically in a slightly different context by MAYNARD SMITH (1968). PROUT (personal communication) has shown that models that incorporate this sort of delay can overshoot equilibrium and cycle in a manner exactly as that shown by MAY (1975) for simpler discrete time models.

If a feed forward effect of density is common, as this study suggests, then it is clear that attempts to measure density sensitivity depend critically on the stage of census. For example, DEBENEDICTIS (1977) estimated fitness from an adult to adult census. His experiment, therefore, included two densities, the one from which the parental females emerged and the one that their offspring create, only one of which entered his analysis.

The demonstration of substantial genetic variation in the magnitude of density sensitivity satisfies, in part, a major assumption of theories of density dependent natural selection. The differential evolution of fitness components in response to density requires genetic variability of the density sensitivity of these components in the first place. Though theories of density-dependent selection predict the outcome of competition between genotypes, variance of density sensitivity in pure culture is clearly a necessary requisite for this sort of evolution, even if it is not sufficient. If two chromosomes show differences in density sensitivity in pure culture, then if placed in competition against one another, the most parsimonious prediction is that these differences would, on the average, be retained. Selection would, therefore, be density dependent. This is meant in no way to deny the possible importance of genotype interactions demonstrated by, for example, LEWONTIN (1955). DEBENEDICTIS (1977) made an attempt to evaluate the relative importance of density and frequency dependence (genotype interaction), and in his experimental system found substantial frequency

dependence and no evidence of density dependence. His work is, however, difficult to evaluate, as it has some methodological problems, one of which was mentioned above. In addition, all the densities he used were very high. If either of the nonlinear density regulating functions listed above are reasonable descriptions, then at high densities (relative to the equilibrium), density dependence will be difficult to detect. Further, the product moment fitness estimator used by DeBenedictis will be spuriously frequency dependent (especially at high densities) if density regulation occurs, unless every individual has exactly the same effect on population growth, independent of genotype. The extent of variability in genotype interactions and a measure of the magnitude of their effects relative to the magnitude of the main effects of density dependence await an unbiased assay of natural populations. That is, just as with density dependence, previous studies of genotype interactions and frequency dependence have demonstrated what is possible, but not what is probable.

There is a suggestion in the data of variation not only in the magnitude of density regulation, but also in the mathematical form that it takes (see Table 4). The issue of the mathematical form of density regulation is important for at least two reasons. First, the three models considered here have qualitatively different stability properties (May 1975); conclusions drawn from any particular model may not be robust with respect to choice of models. As an example, Hassell, Lawton and May (1976) fitted models of single species population growth to many sets of data culled from the literature. They found that, though the models they consider were capable of a rich spectrum of dynamic behavior, parameters estimated from the majority of populations studied predicted only a monotonic return to a single stable equilibrium after perturbation. They concluded that the more complex sorts of behavior must be rare in single-species populations. However, if the simple inverse model is the most reasonable description of density regulation, then in fact the question asked by Hassell *et al.* is moot: the behavior of the inverse model is never other than a monotonic approach to a single stable equilibrium.

The second reason that the mathematical form of density regulation is critical is shown by the theoretical work of Turelli and Petry (1980). They showed that the appropriate choice of models can provide a theoretical counterexample for virtually any prediction about the evolution of life history characteristics. Though the data of this study do not resolve the issue, we clearly must know what forms of density regulation are reasonable descriptions of nature, since the qualitiative properties of different models are so different. Further experimental work is needed to resolve the chaos of models in the literature.

As collected, the data of this study also permit an examination of correlations between the various fitness components and their density sensitivity. At their most basic, theories of the evolution of life history characteristics assume some trade off between performance at, for example high and low densities (see Stearns 1976, for a penetrating discussion). There are two direct and simple methods to examine the data of this study for trade offs. First, traits can be examined, a pair at a time, for cases in which an argument for some trade off can be made. For example, naive considerations of r-K theory suggest that the

value of any component at a low density should be negatively correlated with the value of the same or some other component at high density. Other possible trade offs might be density sensitivity of one component for density sensitivity of another, or for the value of one component with its density sensitivity. Sixteen comparisons of these sorts have been made; in no case is there any evidence for a trade off. Second, the components can be mathematically combined in some more or less arbitrary fashion to get a coefficient related to net fitness. If components are being traded off, this coefficient would be expected to be less variable than the components themselves. Two mathematical combinations of the components, one based on biological arguments and the other arbitrarily assigning equal weights to all components, have been examined. In neither case was the net fitness coefficient noticeably less variable than the components. For a variety of reasons these tests with these data are not very powerful. Again, further experimental work on this problem is clearly necessary.

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